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A CONTRIBUTION TO THE SUBJECT OF ACUTE PLEURISY: ITS PATHOLOGY, ETIOLOGY, SYMPTOMATOLOGY, AND TREATMENT.

BY

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OF NORTHAMPTON, MASS.;
FORMERLY LECTURER ON DISEASES OF CHILDREN IN THE COLLEGE OF PHYSICIANS AND SURGEONS, BOSTON, MASS.

FROM
THE MEDICAL NEWS,
January 21, 1893.

[Reprinted from THE MEDICAL NEWS, January 21, 1893.]

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THE proper classification of an inflammatory affection of a serous membrane, and especially of the pleura, is not an easy task. The terms "acute," "subacute," and "chronic," that have been applied to diseases of that nature, are often subjects of much criticism, no matter what pathologic lesions or symptomatic indications they may be derived from.

In considering acute pleurisy, I shall not hope to unravel these disputed questions, and only trust that I shall not fall into the common error of making them more intricate.

The term "acute" pleurisy indicates an inflammatory condition, more or less general in extent, of the serous membrane lining the pleural cavity, with sero-fibrinous or purulent deposits, primary or secondary in nature, of microbic or other origin, with a certain degree of sharpness of attack, and

[1] Read at the meeting of the New York State Medical Association, held in New York City, November 15, 16, 17, 1892.

in which the height of the disease is attained in a few days, ten at the most, and accompanied by a rise in temperature of greater or less extent. Acute pleurisy may thus be (1) dependent on preëxisting disease or lesions, the development of which may cause sudden intense inflammatory conditions of the pleura, more or less extensive; or (2) it may be an idiopathic disease.

PATHOLOGY.—Hyperemia, or congestion of the bloodvessels within the serous and subserous connective tissue, causing swelling, redness, and edema of the pleura, is the first noticeable lesion of acute idiopathic pleurisy. This congestion may take place in some localized spot and rapidly extend to a greater or less degree; or it may, from the first, present quite a general appearance over the surface of the pleura. Soon the smaller and weaker capillary vessels rupture, producing ecchymotic spots over the membrane, and, at the same time, infiltration of the subserous connective tissue occurs, with a proliferation and detachment of epithelial cells. As the process progresses the pleura is studded with fine granulations upon its surface, in which appear embryonic cells. These tend to organize the newly-formed connective tissue into firm fibrinous bands, which, in old cases, are often found stretching across the pleural cavity, and constitute the so-called neo-membranes. Finally, from the congested serous membrane is poured into the cavity a liquid resembling in all respects the plasma of the blood, except that it is more dilute; the degree of dilution, however, varies with the intensity of the congestion, according to the coagula-

bility of the fibrin in the effused fluid. There are, also, red blood-corpuscles and leukocytes in the liquid; but in simple sero-fibrinous pleurisy the red globules are not in sufficient numbers to cause any marked discoloration; if such occur, the pleurisy is termed "hemorrhagic."

"Inflammation," says M. Germain Sée,[1] "is a struggle for life, and not a destructive process; it is essentially a vital phenomenon eminently reactionary against a morbid agent." He believes that the teachings of microbiology show that inflammation is a physiologic process strongly exaggerated; a general struggle of the organism against microbic invaders. "The first step in this process," he says, "is leukocytosis, or the exaggerated production of white corpuscles in the blood; and the second is the absorption and destruction of microörganisms by these leukocytes, showing the defensive action of the latter; this is called "phagocytosis."

Certain physiologic functions are attributed to the phagocytes:

1. They carry from the albuminates in the intestinal canal material for combustion in the tissues.

2. Owing to their ameboid movements they are capable of transporting to distant parts of the body substances in their vicinity.

3. They possess, under certain conditions, a reproductive function, and a power of collecting themselves in vast quantities in certain localities.

4. They have what is known as a chemotactic function; so that the leukocytes thus play a con-

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[1] Bulletin de l'Académie de Médecine, May 10, 1892, p. 680.

siderable rôle in the morphologic contexture, as well as the chemistry of the body.

The French supporters of the germ-theory of disease believe that when microbes have penetrated the organism, the leukocytes increase; that the conditions that bring them to the blood carry them to the point of excitement, and that it is a chemical property that draws them there. This, they claim, is true of all high-grade inflammations, as pneumonia, but not of the low grade, as malarial fevers.

In pneumonia there is an increase of leukocytes during the height of the disease, but as the fever abates the number of leukocytes diminishes. It is, therefore, held that the real termination of the disease, whether by crisis or lysis, occurs at the time when the leukocytes begin to diminish in the blood.

In acute inflammations there are three stages of development: First, vascular dilatation; second, activity and proliferation of endothelium; third, exudation, with diapedesis of leukocytes. As a consequence of these three stages it is claimed that a great afflux of phagocytes takes place toward the point of attack, both in purulent as well as in catarrhal inflammations; it is less seen in serous varieties, and, perhaps, not at all in infectious processes, the reason for this being that the infectious matters destroy the phagocytic function of the leukocytes, and hence the body has no protecting element against the enemy, and becomes the prey of the microbe. Suppuration is considered no exaggeration of inflammation, but is primarily due to the action of streptococci or staphylococci, and although great numbers of phagocytes may be

found, yet their defensive action is much harassed by their deadly enemy, the pus-germs.

From the foregoing, if we are to accept this doctrine, we may conclude that inflammation is a physiologic process to develop phagocytes for the purpose of antagonizing microbes.

Anatomically the pleura is a great lymphatic sac, contiguous with the arterio-pulmonary system, and naturally derives its serosity from that source.

According to the eminent French teacher, M. Guerin,[1] "pleurisy is nothing else than lymphangitis." "If one injected the pulmonary artery," says Guerin, "with colored liquid, it would be found that the liquid would appear in the polygonal ramifications of the lymphatics of the pleura."

Moreover, he has practised on the cadaver the injection of bullock's blood in the same manner, and finds that he obtains a serosity from the pleura which, if much force is used in the process of injecting, becomes bloody, or red in color. This is on the healthy lung and pleura. If, on the other hand, he injects a colored liquid in a subject that has suffered and died from pleurisy, there will be found no coloring-matter in the meshes of the lymphatics except those that have not been affected by the inflammatory process. If this be true, we can readily see that when from some cause the lymphatics of the pleura become congested and swollen, and the natural channels for the lymph impeded, an edematous condition of the subserous connective tissue will arise, due to forced diapedesis of the

[1] Bulletin de l'Académie de Médecine, April 26, 1892.

serous exudate, and cause the fluid to ooze from all parts into the cavity.

If, then, the lymphatic stoppage is complete enough, and the force behind is strong enough, there will also be more or less diapedesis of red blood-corpuscles, and hence the hemorrhagic pleurisy. If we find leukocytes in the lymphatic exudation, does that not best explain the formation of pus? It has been stated that the serous exudate of acute pleurisy does not differ materially from the plasma of the blood. (The reason for this appears plain if we consider the contiguity of the arterial capillaries with the lymphatics.) If drawn off, it will coagulate, often spontaneously; if beaten or whipped, it will show a deposit of fibrin; and the constituents are the same as the plasma of the blood, except in relative proportion.

It often happens, when one is practising puncture to draw off the effusion of acute pleurisy, that the needle, if small, becomes clogged with fibrinous flocculi. From this fact, it is argued by M. Lancereaux,[1] that these flocculi, and more especially the finer ones, become obstructed in the openings of the lymphatics and produce a mechanical hindrance to the exit of the effused fluid by the thrombus thus formed. When this has occurred to such an extent that it impedes the absorption of liquid beyond the natural time required for the evolution of the disease, he avers, "one must wait for the disintegration and absorption of the clot before any dimin-

[1] Bulletin de l'Acad. de Méd., May 3, 1892, p. 661 *et seq.*

ishing of the quantity of the fluid by nature takes place."

Secondary pleurisies, or those acute attacks from some preëxisting disease which often occur, doubtless present in many cases the pathologic lesions in the pleura upon which such disease depends; but I can see no reason for the opinion of some authors, that every pleurisy is dependent on lesions of pre-existing maladies, and especially tuberculosis.

ETIOLOGY.—In considering the causation of acute pleurisy, one must of necessity admit that there are two general classifications from an etiologic point of view:

a. Those that are not due to any preëxisting disease;

b. Those that are so due.

At the present day there are many and diverse opinions held by eminent medical men as to the factors that produce primary idiopathic pleurisy. There are, indeed, those who would go so far as to say that all pleurisies are tuberculous in origin, and, hence, secondary.

Undoubtedly, many pleurisies deemed primary are in reality dependent on some complicating or preëxisting disease; but to say that a person must be weakened by constitutional maladies in order to become privileged to have pleurisy, seems as if we were carrying the causative factors beyond the point warranted by our pathologic research. I doubt not, if the chests of many persons who to-day are in good health and have never been cognizant of pleurisy were opened, we would find various traces

of old adhesions and other ancient lesions of former pleurisies.

It must, therefore, be admitted, even at the present day of biologic research, that the etiology of acute pleurisy is often obscure: the microbe will not account for all the cases; neither will any other one causative factor. While it is difficult to state with certainty that pleurisy originates in perfectly healthy persons, because latent pathologic lesions cannot be appreciated, yet we know that it does occur in persons who to all appearances, to themselves and others, are in good health. M. Sée[1] maintains that the etiology of acute pleurisy is always microbic; in fact, that it is a bacterial disease; cold is simply a stimulus to the activity and development of the microörganisms.

M. Jaccoud[2] is of the opinion that in the human body many kinds and many thousands of microorganisms live in peace and harmony together so long as the functions are normal, but let a disturbing element arise, such as taking cold, and their physiologic relations become altered, so that they soon are hostile to each other and cause disease. Netter[3] claims that all forms of pleurisy are of microbic origin, but that the microbes producing them are of many different kinds.

M. Bechamp[4] says: "Microbes do not have so much importance in acute pleurisy as some would have them." He is certain that pleurisy may exist

[1] Bulletin de l'Académie de Médecine, May 10, 1892.
[2] Ibid., June 7, 1892.
[3] British Medical Journal, June 7, 1892, p. 793.
[4] Bulletin de l'Académie de Médecine, May 17, 1892, p. 758.

independently of tuberculosis, from the fact that at the age of thirty he was seized with acute pleurisy, and after the usual bleeding, blistering, and purging, he now, at the age of seventy-six, is still alive, and has not developed tuberculosis. In support of this, both he and Dieulafoy state that, in many cases, persons live ten, fifteen, and more years after the operation of thoracentesis, and do not develop tuberculosis.

That exposure to cold has a tendency to excite inflammations, and with them acute pleurisy, is no doubt true. Whether its influence is exerted through the nerve-centers, so as to directly cause pleurisy, or whether it acts simply as a stimulus to organized germs through whose activity the disease originates, is still a much-mooted question. M. Tresbot[1] does not doubt that acute pleurisy in horses is the direct result of an exposure to cold, especially when, after a long, hard drive the animal is allowed to stand unprotected and exposed to a chilling wind. He says that "ordinarily there is nothing in common between sero-fibrinous pleurisy in the horse and tuberculosis;" and also that "it is impossible to class sero-fibrinous pleurisy in the horse with an eruptive fever, or, indeed, with any periodic disease." On the other hand, M. Lancereaux[2] asserts: "Pleurisy should be rightly classed among the infectious maladies, and exposure to cold is nothing but an occasional exciting cause, while the action of the infecting agent still escapes us."

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[1] Bulletin de l'Académie de Médecine, May 17, 1892.

[2] Ibid., May 3, 1892.

If we should accept the pathologic views of M. Guerin,[1] we might easily explain the causation of the congestion of the bloodvessels by peripheral irritation, and reflex action of the vasomotor nerves from exposure to cold. This, however, does not explain why such excitement should be directed particularly to the pleura.

In a paper on "The Cause of Syncope in Pleurisy," M. La Borde[2] has illustrated by experiment on animals that it is possible to produce a sero-fibrinous pleurisy in a few hours by the action of cantharidin injected into the blood. This leads me to ask the question, Is the causation of acute primary pleurisy ever, in a measure, governed by the ingestion of certain articles of food, taken just previously to an exposure to cold, *i. e.*, is the combination of the two forces sufficient to direct the action of inflammation toward the pleura?

The cause of acute purulent pleurisy is probably of microbic nature. Purulent pleurisies either begin as such, or are secondary to other diseases. Age and debility have much to do with the formation of pus, the young and aged being more susceptible to empyema. It is doubtful if simple sero-fibrinous pleurisies are ever transformed into the purulent form without the aid of outside interference.

Secondary pleurisies occur from a variety of causes, mostly from diseases microbic in origin.

While it may be rare to have pneumonia occur without some localized extension of inflammation

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[1] Loc. cit.

[2] Bulletin de l'Académie de Médecine, May 17, 1892, p. 709.

to the pleura, it is doubtful if a general acute pleurisy, secondary to pneumonia, can be found without numerous pneumonia-cocci. In the same manner, the causation of pleurisy during an attack of typhoid fever, influenza, malarial fever, rheumatism, and kindred diseases, is undoubtedly due to the same influences that govern the coëxisting disease.

It is well known that pleurisy of an acute type may be secondary to tuberculosis; but there is considerable difference of opinion as to the proportion of cases arising from this source. M. Sée[1] claims that 68 per cent. of all pleurisies are due to this cause. This seems high, or else persons radically recover from tuberculous pleurisy more often than from any other form of tuberculosis. Dr. G. G. Sears[2] reports four hundred and fifty cases of pleurisy, of which 39 per cent. developed tuberculous disease. Others state that not more than 20 per cent. of pleurisies are tuberculous in origin. The differences, probably, lie in the particular train of cases various observers have met.

There is, however, no doubt that a certain number of cases of tuberculosis have their initial symptom manifested by an attack of acute pleurisy.

Symptomatology.—M. Lancereaux and other French observers believe acute pleurisy to be a well-defined cyclic malady,[3] because its lesions are always found unchangeable, and its evolution is as constant as that of pneumonia or of typhoid fever. It presents regular pathologic changes after each seventh day,

[1] Bulletin de l'Académie de Médecine, April 19, 1892.

[2] Boston Medical and Surgical Journal, Feb. 25, 1892.

[3] Bulletin de l'Académie de Médecine, May 3, 1892.

and this fact allows a classification of the disease into three periods of evolution.

First: Seven days represent the time occupied for the increase of the pathologic lesions, at the end of which the effusion reaches the limit of its advance.

Second: The next seven days represent the time during which the pathologic lesions appear to remain stationary, and the effusion has not undergone any perceptible alteration in amount.

Third: From the fifteenth to the twenty-first day of the disease there takes place an absorption of the inflammatory products, including the effusion. These divisions, of course, cannot be made absolute, because individual cases differ among themselves; thus we all know that fluid-accumulations often completely fill the pleural cavity before the expiration of the first seven days, and when withdrawn reaccumulate; however, we may confidently expect at the end of the first period that in uncomplicated cases there will be no further effusion of fluid.

Each of these three stages has its respective symptoms, but they pass from one to the other without any pronounced expression of change.

In a majority of cases the first period of acute pleurisy is ushered in with a chill, announcing a greater or less rise of temperature, and accompanied with more or less pain in the affected side.

The pyrexia will generally determine the intensity of the inflammation; it seldom rises above 102° or 103°, unless the pleurisy is secondary to some other disease. Pain, which appears with the pyrexia, may begin as soreness in a circumscribed spot and

progressively increase to an unbearable intensity; or it may at first be sharp and lancinating, at or below the nipple, from which point it becomes more or less diffused. The pain is increased on motion, and, for this reason, respiratory efforts are, so far as possible, restrained. A dry, hacking cough is generally present, but the pain it occasions calls for efforts at its suppression. After a few hours, when the effusion appears, the pain is moderated and gradually disappears, or its intensity is greatly diminished. The pulse is increased in frequency and generally firm, the rate varying between 100 and 120. As the disease progresses, dyspnea is often developed, and this may be due to several different causes. It may indicate abundant effusion, and be due to compression of the lung or displacement of the heart; it is most commonly a result of one of these. Again, it may be due to congestion of the lungs independently of compression. Edema of the lungs, capillary bronchitis, and rheumatic difficulties sometimes occasion dypsnea when very little fluid can be found in the cavity. Encysted pleurisies occasionally cause pain and dypsnea. Irregularity of the pulse and cyanosis are grave symptoms, largely dependent on displacement or weakness of the heart. Syncope may also be occasioned in various ways; the reflex action of pain is one of its chief causes, as we may often produce it artificially by electrifying some peripheral sensory nerve. Again, when the fluid fills the pleural cavity and is suddenly drawn entirely away, the reaction that follows may cause syncope.

Physical Signs.—These vary with the quantity

rather than quality of the effusion. In the early stages of the disease they often establish the diagnosis, and in the later stages they determine the amount of liquid in the cavity of the chest. On inspection there is in the early stages restricted motion of the affected side, dependent at first on the amount of pain, and later on the accumulated fluid. The comparison with the sound side is very striking. In left-sided effusions there may be displacement of the heart-beat to the right of the normal, and when large effusions occur on the right side, displacement is sometimes to the left. On palpation, vocal fremitus is lost over a collection of fluid, and if the effusion is large on the right side, the liver may be felt to be displaced. On percussion, we may early find dulness over the lower posterior portions of the chest; this dulness gives place to flatness as the effusion appears and advances; above the fluid the percussion-note remains dull, while at its level, and below, it becomes flat. It will be remembered that the level of the fluid is not a hydrostatic one; but from the elasticity of the lung the level assumes a shape resembling the letter S. Over the sound lung the vesicular resonance is more or less exaggerated. Dulness is likely to remain for a considerable time after the effusion has been absorbed. On auscultation, the respiratory murmur is enfeebled or absent, for the same reasons and in the same progressive manner as the loss of resonance on percussion occurs. Friction-sounds are sometimes heard at the beginning of the disease, but are more common at the close of the third stage. Some observers have indicated that the transmission of the whispered

voice, or its absence, has direct relation to the diagnosis of serous or purulent effusions, but this is doubted by others.

No lengthy discussion of the physical signs of acute pleurisy is necessary here; these do not differ from their description as set forth in our modern text-books.

TREATMENT.—In considering the treatment of acute pleurisy we must recall the classification of its etiology, viz.:

First: Those cases that are dependent on some other disease for their cause, whether influenced by microbes or not, and hence are secondary pleurisies.

Second: Those cases that are of spontaneous origin, considered idiopathic, and hence are called primary pleurisies.

Physicians may likewise be divided into classes: those who consider pleurisy to be always a microbic disease, and those who do not.

In a recent discussion on this subject the eminent French author, Hardy,[1] said that acute pleurisy of sero-fibrinous nature was no better treated to-day than it was fifty years ago, and, except in purulent forms, no better results were obtained now than then, the death-rate at present being 10 per cent., the same as in the days of our forefathers. This statement may be astonishing to some of us who have been taught to look upon acute pleurisy as a not very fatal disease; however, some statistics would seem to bear out this opinion. Perhaps it would, therefore, be well to consider the modern methods of

[1] Bulletin de l'Académie de Médecine, May 1, 1892, p. 776.

treatment and then compare them with those practised in the early part of the century.

To undertake the consideration of all the medications for pleurisy that have been launched upon us during the past few years would take more time and space than would be profitable for me to employ; suffice it to say that a majority of them have passed into disuse.

The modern medical treatment of acute pleurisy is by the following class of agents: First, by antiseptics, to combat microörganisms; second, by antipyretics, to combat fever; third, by evacuants, to eliminate the fluid.

Dr. Charles Talamon[1] has recently called atten tion to the action of sodium salicylate in pleuritic effusions, claiming for it the power of promoting rapid absorption of the fluid. He thinks it has a direct action on the inflamed pleura, because by the experiments of Rosenbach it is proved that the salicylates may be found in the serous cavities of the body after their ingestion by the mouth in doses of from ten to twenty grains. Whether the beneficial action on the fluid is due to the antiseptic nature of the agent, he does not state, and whether it is due to this or its diuretic action is still an open question. That sodium salicylate may be of use when the pleurisy is secondary to rheumatism there can be no doubt, but in the primary form to depend on its success as a germicide would be hazardous. However, the salicylates may be employed as antipyretics as advantageously as other remedies; they

[1] New York Medical Journal, January 2, 1892.

certainly combine the indications for an antiseptic, antipyretic, and diuretic.

The practice of injecting a solution of salicylic acid or other antiseptic into the pleural cavity to combat microbes in the effusion has been suggested by some, but the treatment seems harsh and uncalled for, unless employed in connection with surgical methods for the treatment of empyemas.

Antipyretics in acute pleurisy are only indicated when the fever rises to 101° or over, and as the fever seldom attains that height for any length of time their use is greatly modified.

Quinine may be advantageously employed in pleurisy depending on malarial poisoning, and during convalescence, as a tonic. Antipyrin, or the other coal-tar derivatives, may be useful in cases accompanied by hyperpyrexia, but none of these measures is calculated to reach the cause of the disease, or to modify its pathology.

The evacuants are administered in acute pleurisy with a view to reduce the amount of effusion after its accumulation.

Under this head, the diuretics play the most important part. Digitalis may support a weak heart, but its action in reducing a pleuritic effusion is small. Milk is often used as a diuretic, but its influence over fluid in this disease is doubtful; while as a food it ought not to be neglected.

The action of purgatives, drastic or saline, and of sudorifics, with a view to reduce the quantity of liquid in the chest, is of no value; moreover, they are often dangerous.

The pleuritic effusion is not really a question of

hydropsy; the liquid of general ascites furnishes a chemical analysis quite different from the effusion of pleurisy; the latter is not simply a serum from the blood, but blood-plasma.

In a recent paper on "The Treatment of Pleuritic Effusion," M. Sée[1] draws the following conclusions: "Antiseptics, diuretics, sudorifics, and purgatives, drastic or saline, have no kind of action on the effusion. Milk, which is a powerful diuretic, has no value here, except as a food. No one of these microbic diseases derives the least benefit from venesection. All aggravate the onset of the disease. Expectation is the only rational method of treatment, for sero-fibrinous pleurisy regularly passes through its different phases in two or three weeks, and up to that point all medication is useless." In a recent paper on this subject, M. Lancereaux[2] says: "There is no more use in trying to ward off pleuritic fever than to ward off pneumonia or typhoid fever; however, it is necessary, relying on our pathologic knowledge of the lesions of acute pleurisy, to draw attention to the coagulation in the lymphatic system, and strive to remove it; while we may not succeed, it is the best practice to try."

In discussing the subject, M. Guerin[3] says: "This idea of pleurisy being a simple lymphangitis also gives a clear explanation of the sudden reabsorption of fluid in some cases that for a long time have proved refractory to all treatment. It indicates that the coagulum in the absorbent vessels has become

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[1] Bulletin de l'Académie de Médecine, April 19, 1892.
[2] Ibid., May 2, 1892. [3] Ibid., April 26, 1892.

reabsorbed and makes it possible for the fluid to be taken up; we should therefore attempt the prompt moderation of the lymphangitis."

Of late, in the excitement of bacteriologic investigations, the profession had been content to disregard the teachings of our ancestors, and the good old methods in vogue at their time. We have regarded these diseases as cyclic in nature; maladies that must run their regular course in spite of all abortive treatment; sicknesses that are caused by microorganisms over which we have no control; hence, we must fold our hands and content ourselves with relieving of pain, until such time as the particular germ has loosened his besieging grasp.

This is the "expectant treatment of to-day, and this is the treatment eminent authorities hold out to us for acute pleurisy." "The expectant treatment," says Peter,[1] "is the do-nothing treatment, and this is what many bacteriologists practise. No physician has a right to practise inactivity in these cases when so much can be done toward curing pleurisy if energetic treatment is begun early."

To allow a patient to become weakened by pain and suffering, because pleurisy is a cyclic disease and should terminate spontaneously in two or three weeks, or, because the pathologic conditions are such that, by deferring active measures, one may with impunity puncture the chest and draw off a liter of fluid, is treating the wrong end of the disease. A physician should strive to diminish the in-

[1] Bulletin de l'Académie de Médecine, April 26, 1892.

tensity of a malady, if, indeed, he cannot shorten its duration.

What are the indications for treatment in acute pleurisy?

Briefly, they are as follows: 1. The relief of pain. 2. The reduction of fever. 3. The arrest of effusion. All of which depends on treating the cause.

It was of these indications our ancestors had a rare knowledge when they applied bleeding and localized vesications; when practised, these at once relieved the pain, reduced the fever, and there was little or no serous effusion found in their cases; moreover, if in this they were not entirely successful, the harshness of the sickness was notably diminished. By these methods the pathologic lesions were affected, and the cause of the pain and fever reached. In 1819, Laennec said: "If a plethoric subject had pleurisy, he required bleeding."

While it is undoubtedly true that the pendulum of exsanguination in inflammatory diseases oscillated too far in the early part of this century, and to the detriment of many, is it not also true that at the present time it has swung too far the other way, and "expectant treatment" is employed to the detriment of many? I believe the antiphlogistic treatment is indicated in just as many cases of acute pleurisy now, as in the days of our forefathers. That the treatment may be carried too far I do not deny; the judicious use of it at the right time is the essential point. Pain is almost wholly relieved by the revulsive methods in a short time; this probably being accomplished by the removal of the congestion and its baneful influences, if revulsion is prac-

tised early. At the present time, and in some form, opium is employed for the relief of pain; but does it lessen the congestion causing the pain, or simply dull the sensibility to the pain which is the result of the congestion? Revulsion lowers the fever, for which we now employ antipyretics, but will the antipyretic drugs alter the pathologic condition within the pleural cavity?

Farther on in this disease we often employ puncture of the chest to draw off the fluid, but have we not allowed the malady to reach that point by an early neglect to treat the cause efficiently, and thus converted what should have been a medical case into a surgical one? This certainly is to be deplored. Peter[1] goes so far as to say, that "By revulsive methods, early employed, one is able to prevent the serous effusion in many cases; to arrest it, if the secretion has begun to form, and finally to absorb it, if a small amount has collected." Andral and Bouilland[2] cite thirty cases in one year, treated antiphlogistically, with one death—percentage of mortality of three. Of seventy cases reported[3] in 1891, treated after the modern methods, there were seven deaths—a death-rate of 10 per cent. from acute pleurisy.

How should revulsion be employed in these cases?

Bouilland used to bleed twice and afterward apply wet cups and a large blister. This seems too energetic; yet I have recently been told by an elderly physician of my neighborhood that he once bled a woman with puerperal peritonitis until she

[1] Bulletin de l'Académie de Médecine, April 26, 1892.
[2] Ibid. [3] Ibid.

fainted, and she had no more symptoms of the disease, and was soon about. One might employ wet cups and vesication more mildly, with equal success. "What is remarkable in these cases," says Peter,[1] "is the toleration of the system to these large bleedings, the rapidity with which the pain ceases and the local symptoms of pleurisy diminish." On the other hand, "Bleeding," says Sée,[2] "after doing so much harm, is springing up again insidiously in many diseases; it has no theoretic basis, and is little better than empiricism."

Can we learn anything from Nature as to the treatment of acute pleurisy?

There is pain from irritation of the intercostal nerves, due to the evolution of pathologic processes. Nature, in consequence of this inflammatory action, evolves a fluid which acts as a sedative to the injured nerves, and, little by little, the pain subsides. The fluid comes from the blood. This fluid is found to be exactly like the plasma of the blood, except in proportion; is it, therefore, not natural to assume that Nature bleeds her patient in the early stage of pleurisy? To be sure it may be the result of a congested condition, and, hence, one of pathology rather than one of treatment; nevertheless it depletes the vascular system, which, if we anticipate Nature by doing ourselves, we thereby arrest the pathologic condition and relieve Nature.

Regarding the application of blisters, there is a diversity of opinion, although it is generally held that they are useful adjuvants to scarification.

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[1] Bulletin de l'Académie de Médecine, April 26, 1892.

[2] Ibid., April 19, 1892, p. 97.

Metschnikoff[1] advances the idea that cantharides possesses an anti-bacterial tendency, and produces leukocytes which act as phagocytes, and proceed to destroy or change the character of the microbe. He would, therefore, inject a solution of cantharidin subcutaneously. Tresbot[2] has no doubt that a cantharides blister is of great value in the treatment of the pleurisy of horses. La Borde,[3] while advocating the use of cantharides blisters, calls attention to the fact that this agent is a poison, and capable of producing inflammation of the lung, bladder, or other viscus, and cautions against its use. There has been some belief that cantharides predisposes to the transforming of serous effusions into purulent ones, and especially in tuberculous patients. Potain denies this,[4] or the possibility of its happening, and maintains that a purulent effusion always starts as such. Counter-irritation may also be effected by the tincture of iodine painted on the surface of the affected side. Little in addition can be said of the treatment of purulent pleurisies prior to surgical interference, which is almost always required.

There is no positive way to differentiate them from the serous varieties, except by explorative puncture, but the indiscriminate use of the exploring-needle, ten or a dozen times, as recommended by some,[5] in search for pus, is to be condemned, even if no harm should chance to arise.

It is in childhood and old age that purulent pleu-

[1] Bulletin de l'Académie de Médecine, April 19, 1892, p. 602.
[2] Ibid., May 17, 1892. [3] Ibid. [4] Ibid.
[5] Archives of Pediatrics, May, 1892, p. 353.

risy is most likely to occur. Why this is so, unless from a weaker state than is present in adults, is not easily determined; but in the treatment of this affection in children, this fact is to be borne in mind.

Thoracentesis.—This operation, though always to be deplored, is often urgent and often useful. A discussion of its history, which may be found at length in *Pepper's System of Medicine*, is unnecessary in this place. Of late there has been much criticism for and against the operation by eminent authorities. That aspiration, as first practised by Bowditch, and later elaborated by Dieulafoy and others, is a simple, harmless operation, there can be no doubt; results will bear this statement out. All that is essential to the safety of the operation is thoroughly aseptic instruments, especially the aspirating-needle, and also some little skill in manipulation.

Thoracentesis, as practised by the majority of general practitioners, with any kind of trocar, which may have been used by them to open some abscess-cavity, or even with an aspirating apparatus that has not been perfectly cleansed, is a very dangerous operation.

It has been held by some that after the operation has been performed two or three times it produces a transformation of a sero-fibrinous effusion into a purulent one. This may be so; I do not deny that it often happens, but it is the operator and his unclean instruments that are at fault, rather than the effect of a puncture of the pleura; this is the reason we

see one operator successful where another meets with failures.

As a rule, aspiration should not be performed in simple sero-fibrinous pleurisy until after the third week of the disease; and then only as the fluid tends to remain stationary and unabsorbed, unless there is urgent need of interference to save life before that time. If the cause of the non-absorption of the fluid is (according to the views of Lancereaux) a stoppage of the lymphatics of the pleura by the formation of fibrinous thrombi in their orifices, and if we must wait until a disintegration of the clot takes place before the fluid will be absorbed, then no amount of aspiration will hasten the process of natural absorption until that time has expired. Moreover, the drawing off of the fluid will in many cases only tend to its reaccumulation up to the point it previously reached, because, according to his theory, if the fluid remains stationary to a given level or height within the cavity of the chest, there must be stoppage of all the lymph-spaces below that level; hence, no absorption is possible. If, then, we remove a part or the whole of the fluid, we do not necessarily remove the stoppage, and the reaccumulating fluid will in time reach its former height.

On the other hand, if, before practising thoracentesis, we wait until the disease has reached that period when we may expect these clots to be disintegrating and being taken up by the system, we then may be of some service to Nature in hastening absorption. This period is at about the end of the twenty-first day of the disease. However, there are times previous to this period, when life is threatened by

the accumulation of fluid in the pleural cavity to such an extent that it compresses vital organs. When this occurs, it becomes necessary to draw off a certain amount of fluid by aspiration, in order to relieve distressing symptoms.

By what symptoms can we know that this danger is imminent?

When the fluid-accumulation has progressed to the extreme degree, there is dyspnea, from compression of the lung; more or less cyanosis, if the heart be displaced; flatness on percussion over most of the affected side, and sometimes accompanied with bulging of the intercostal spaces. However, if we wait for these symptoms to appear, there are strong dangers of being too late to render the assistance necessary.

There is in these cases no infallible sign by which we may discover the best time to operate.

Dyspnea, as we know, may be due to other causes than compression of the lung or displacement of the heart, and consequent twisting of the large arteries by fluid. Cyanosis and syncope are signs that may arise from hearts weakened by excess of pain, with little mechanical interference from an effusion into the pleura.

The most reliable test is by percussion; as by it we may arrive at an approximate estimate of the quantity of fluid contained in the pleural cavity. When the line of flatness has reached the second rib on either side of the chest, the left or right, Dieulafoy estimates the quantity to be about 2000 grams, and states it is time to operate. If, with this percussion-sign, there is dyspnea and some cyanosis,

it is time to hasten the operation; as, while it is not right to puncture the chest too early, it is also hazardous to postpone the operation too long.

If, as often happens after early operative interference, the fluid should reaccumulate more or less rapidly, and the symptoms of distress reappear, a second operation would be imperative, and probably later on others would necessarily follow.

Only so much of the fluid should be removed in these cases as will render the patient more comfortable from the distressing symptoms, or remove any danger of immediate collapse. Sudden death has followed the removal of the entire amount of effusion at once, death being due to the congestion occurring from the sudden return of the compressed and distorted viscus to its normal position. A symptom of this danger is said to be the albuminous expectoration observed in these cases. The fluid should therefore be withdrawn gradually through a fine needle, and not more than a third, or perhaps a half, of the total quantity of fluid in the chest-cavity be removed at once. It is better to perform the operation several times in this manner than to have a fatal issue from the evacuation of too great a quantity of liquid at once.

In purulent pleurisies no time should be lost in evacuating the pus, observing the same precautions necessary, if by aspiration, as in serous effusions. In children with purulent pleurisies, repeated aspirations are advisable before resorting to more severe surgical methods; but in adults, if a reaccumulation of pus occurs after one aspiration, it is usually better practice to treat the empyema as one would an

abscess-cavity and establish a system of free drainage. As these several methods of drainage come strictly under the head of chronic pleurisy, I shall not occupy more time here with a discussion of them.

Finally, I wish here to suggest a method of treatment for pleuritic effusions that I must frankly state is at present a simple theory, because I have not had the time or opportunity to clinically test its value. It is the employment of electrolysis to cause absorption of pleuritic effusions, based on the same theories as when it is used in serous effusions elsewhere. In a word, the operation might be called "electrocentesis." I have not found that any literature on the subject has ever been published—or ever ought to be published; however, electrolysis, as we know, has been employed quite commonly in serous effusions of other localities, such as cysts, hydrocele, tumors, etc., with great benefit in many instances. Why should it not be used with benefit in the serous effusion of pleurisy?

Electricity is used to hasten the absorption of fluid in cysts; first, by its power to chemically transform the watery into gaseous elements; second, by its direct stimulating influence on the lining membrane of the sac.

Is there any reason to expect any different results from the use of electricity, in a similar manner, in pleuritic effusions?

The technique of the operation recommended includes the use of the electro-puncture needle, thrust into the effusion; a clay electrode attached to the negative pole of a galvanic battery, and placed on the outside of the chest-wall. A current of a strength of

from 30 to 50 milliampères, and perhaps more, could be safely passed through the fluid in this manner. Care should be taken that the needle be not thrust farther than just into the fluid, so that we get only electric action on the effusion and the costal pleura, otherwise we might electrify some vital organ in a manner not pleasing. From this application of electrolysis we might reasonably expect more or less coagulation of fibrinous matter and absorption of the fluid portions of the effusion. This we might expect to be in proportion to the strength of the current, and the length of time occupied in allowing the current to pass through.

The class of cases this method of treatment would probably benefit would be those in which thoracentesis for any reason could not safely be performed; those in which there has been repeated reaccumulation of fluid after aspiration, especially those of chronic tendencies; and, finally, those of secondary nature, particularly the tuberculous.

All antiseptic precautions usually necessary in any such operation should, of course, be observed.